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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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Petrina S Hsi
HYSEQ Inc
670 Almanor Avenue
Sunnyvale, CA 94086

EXAMINER
SOUAYA, JEHANNE E

ART UNIT	PAPER NUMBER
1634	

DATE MAILED: 08/04/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/545,283	BOYLE ET AL.	
	Examiner	Art Unit	
	Jehanne E Souaya	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *09 April 2003* .

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *10,11,20 and 31* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *10,11,20 and 31* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

1. Currently, claims 10, 11, 20, and 31 are pending in the instant application. The amendments and arguments have been thoroughly reviewed but are deemed insufficient to place this application in condition for allowance. Any rejections not reiterated are hereby withdrawn. The following rejections are reiterated. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow. This action is FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Maintained Rejections

Claim Rejections - 35 USC § 101

3. Claims 10-11, 20, and 31 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific or substantial asserted utility or a well established utility.

The claims are drawn to isolated polypeptides comprising the amino acid sequence of SEQ ID NOS 4 or 6, to compositions and kits comprising these polypeptides, to an isolated polypeptide comprising an amino acid which is 99% identical to the amino acid sequence of SEQ ID NO 4 or 6, and to a polypeptide encoded by a polynucleotide comprising the sequence of SEQ ID NO 3. The specification teaches that SEQ ID NO 4 is a C-type lectin receptor like polypeptide. SEQ ID NO 4 corresponds to the amino acid sequence encoded by the nucleic acid of SEQ ID NO 3. The specification teaches that SEQ ID NO 6 is the extracellular portion of

SEQ ID NO 4 (see p. 4, lines 28). The specification further teaches that a predicted N-linked glycosylation site is encoded between residues 110 and 112 (Arg His Trp) of SEQ ID NO 4 (p. 4, lines 29-30). The specification, however does not teach the activity or biological function of SEQ ID NOS 3, 4, or 6. At page 4, line 28, the specification asserts that SEQ ID NO 6 is useful on its own as a soluble protein, but does not disclose what this use is, teaching only that this can be confirmed by expression in mammalian cells and sequencing of cleaved product.

The specification asserts the following uses for the polypeptides. At page 7, lines 23-29, the specification teaches that the polypeptides can be used a) to generate an antibody that specifically binds the polypeptide, b) as molecular weight markers, and c) as food supplements. The specification further asserts that the polypeptide can be used to prevent, treat, or ameliorate a medical condition (sentence bridging pages 7 and 8, and page 8 first para) which involve aberrant protein expression or biological activity. The specification asserts that the polypeptides of the invention having C-type lectin receptor activity are useful for prophylaxis or treatment of disorders or diseases caused by or involving allergic reactions, inflammation, sepsis, Alzheimer's disease or other nervous system disorders, bone development, and wound healing (p. 8, lines 26-30). At page 45, lines 5-10, the specification asserts that the polypeptides can also be used in assays to determine biological activity, to raise antibodies or to elicit an immune response, as a reagent in assays designed to quantitatively determine levels of the protein in biological fluids, and to isolate correlative receptors or ligands. The claimed polypeptides, however, are not supported by a specific asserted utility because the disclosed uses of the polypeptides are non specific uses that are applicable to polypeptides in general and not particular or specific to the polypeptide being claimed. It is noted that the specification asserts that SEQ ID NO 4 may

function as a shed receptor, however the specification has not demonstrated such nor is this use specific for SEQ ID NO 4 as a number of other receptors have such a function including IL-2R, TNF-alpha receptor, EPCR (endothelial cell protein receptor) and peritoneal macrophage Fc gamma receptor. The fact that a receptor may be shed does not make clear or apparent the function or specificity of the receptor, nor does it identify the ligand for the receptor.

Further, the claimed polypeptides are not supported by a substantial utility because no substantial utility has been established for the claimed subject matter. For example, a polypeptide can be used to obtain an antibody. The antibody could then be used in conducting research to functionally isolate the protein. The need for such research clearly indicates that the protein and/or its function is not disclosed as to a currently available or substantial utility. A starting material that can only be used to produce a final product does not have substantial asserted utility in those instances where the final product is not supported by a specific and substantial utility. In this case, none of the antibodies that are to be produced as final products resulting from processes involving claimed polypeptides have specific and substantial utilities. The research contemplated by applicant(s) to characterize potential protein products, especially their biological activities, does not constitute a specific and substantial utility. Identifying and studying the properties of a protein itself or the mechanisms in which the protein is involved does not define a "real world" context of use. Similarly, the other listed and asserted utilities as summarized above or in the instant specification are neither substantial nor specific due to being generic in nature and applicable to a myriad of such compounds. Note, because the claimed invention is not supported by a specific and substantial asserted utility for the reasons set forth above, credibility of the utility has not been assessed.

It is noted that the specification teaches that SEQ ID NO 4 has 39% identity to "mouse macrophage C-type lectin" over amino acids 18-232 of SEQ ID NO 4, 49% identity to "dendritic cell immunoreceptor" and "DDB27" (which appear to be the same protein) over amino acids 39-227 of SEQ ID NO 4, and 44% identity to "mouse C-type" over amino acids 16 to 225 of SEQ ID NO 4 (p. 4). The specification further asserts the C-type lectin receptor-like proteins of the invention belong to the same family as C-type lectin receptor, mannose-binding lectins, mammon-binding lectins, and dendritic cell immunoreceptors and therefore have similar activity to these C-type lectin receptor proteins. C-type lectin receptors, however, belong to a large family of proteins exhibiting different structures and functions, such that an analysis based solely on homology or membership in a broad family does not identify the ligand or biological activity or function of SEQ ID NO 4. Akimoto et al teach (Akimoto, Y, et al. Prog. Histochem. Cytochem. 1998, vol. 33, pp 1-92) that C-type lectins are a family of lectins that have a common type of carbohydrate recognition domain (CRD), however they perform diverse biological functions including clearance of molecules from blood circulation (hepatocyte asialoglycoprotein receptors), internalization of foreign and self derived materials, (alveolar macrophage lectin), role in humoral self defense mechanisms (collectins), cell-cell adhesion (selectins), and transmembrane signaling to cells (natural killer cell receptors) (p. 12, section 2.2). With regard to the CRD, Drickamer (Curr. Opin. Struct. Biol., 1999, vol. 9, pp 585-590) teaches that evidence in the art suggests that many protein modules containing part or all of the C-type CRD motif serve functions other than saccharide recognition, and that it is appropriate to consider this motif a characteristic of C-type lectin-like domains to reflect their similarity to CRDs of C-type lectins without necessarily implying common function (p. 585, col. 1, para 2). Figure 3 of

Akimoto et al illustrates the differences in structural organization of C-type lectins, and table 3, teaches the variety of different ligands and sugars for which different C-type lectins exhibit specificity. This wide range of sugars and ligands include galactose, N-acetylgalactoseamine (GalNAc), glucose, fucose, N-acetylglucosamine (GluNAc), mannose, sulfated polysaccharides, and IgE for example. It is further noted that the specification asserts that SEQ ID NO 4 has similar activity to different types of C-type lectin receptors, including mannose binding lectins which belong to the collectin subfamily, while the "dendritic cell immunoreceptor" and "mouse macrophage C-type lectin", which the specification teaches a certain % identity to SEQ ID NO: 4, appear to belong to the type II receptors. From the teachings of Akimoto, however, it is apparent that assignment of SEQ ID NO 4 to a particular subfamily does not make apparent the function or specificity of SEQ ID NO 4 as table 3 shows that different type II receptors have different specificities and bind different ligands. For example human H1 binds galactose and N-acetylgalactoseamine while CD23 binds IgE.

Furthermore, with regard to the alignment of SEQ ID NO 4 with "mouse macrophage C-type lectin" and "dendritic cell immunoreceptor", for example, the art does not teach the function or specificity for either receptor. Balch et al (JBC, 1998, vol. 273, pp 18656-18664) teaches that comparative sequence analysis suggests that "mouse macrophage C-type lectin" (referenced as mMCL by Balch) has carbohydrate binding capabilities, but that little can be postulated about the ability or specificity of mMCL from its protein sequence alone because even within a relatively small, conserved domain, binding specificity can be altered with the mutation of only one or two amino acids (see Iobst and Drickamer, JBC, 1994, vol. 269, pp 15512-15519). Balch further teaches that some molecules containing C-type lectin domains have been shown to bind

peptide sequences such that this versatility makes predicting putative ligands for this type of lectin domain difficult, and that for mMCL this task is even more challenging because a serine rather than the typically conserved proline separates the two critical sugar binding residues corresponding to Glu-185 and Asn-187 (p. 18662, paragraph bridging col. 1 and 2). Bates et al (J. Immunol., 1999, vol. 162, pp 1973-1983) teach that dendritic cell immunoreceptor (DCIR) is a type II membrane glycoprotein (abstract) and that it is of potential importance in regulation of dendritic cell function, however it's function or activity in such regulation is not taught. Bates teaches that the Ca2+ ligating residues are well conserved in DCIR, displaying closest homology with hepatic ASG-PR, but that localization of the gene on chromosome 17 could suggest that DCIR represents an evolutionary intermediate between the NK cell receptors and the hepatic lectins [different type II lectins - see Akimoto et al] and that the cytoplasmic domain of DCIR contains one ITIM motif which is present in the cytoplasmic tail of C-type lectin like molecules expressed by NK cells (p. 1979, col. 2, last two sentences of last full para; and bridging para pp 1979-1980). A sequence search of SEQ ID NO 4 revealed 43.5 % identity to dectin 2, a C-type lectin, however studies showed that a his-dectin 2 fusion protein failed to exhibit specific binding to mannose, fucose, lactose, GluNAc or GalNAc (Ariizumi et al, JBC, 2000, vol. 275, pp 11957-11963; p. 11960, col. 2, last full para.). Therefore the indicated % identity and similarity to C-type lectin receptors would not indicate to one of skill a specific or substantial utility for the claimed polypeptides. While it is credible that SEQ ID NO 4 belongs to the C-type lectin receptor family, the prediction of putative domains does not provide the artisan with a "real world" use for the claimed polypeptides. The specification does not teach what the biological activity or function of SEQ ID NO 4 is, nor does it demonstrate which diseases it is associated

with or would be used to treat. Therefore, the citation of sequence similarity results in an unpredictable and therefore unreliable correspondence between the claimed biomolecule and the indicated similar biomolecules and therefore lacks support regarding utility. Further experimentation would be required of the skilled artisan to determine a use for the polypeptides of the claimed invention. As noted by Brenner v. Manson, 383 US 519, 535-536 (1996), "Congress intended that no patents be granted on a chemical compound whose sole "utility" consists of its potential role as an object of use - testing... a patent is not a hunting license. It is not a reward for the search, but compensation for its successful conclusion."

Claim Rejections - 35 USC § 112

Enablement

4. Claims 10-11, 20, and 31 are also rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a specific or substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention. The teachings in the specification are set forth above (section 13). Given that the art teaches the unpredictability of determining function and specificity with regard to C-type lectins based on homology analysis, and that the specification does not teach the activity or function of the claimed molecules, the skilled artisan would have to perform trial and error to determine the function and activity of the claimed molecules, the results of which are unpredictable, thus constituting undue experimentation.

Response to Arguments

4. The response traverses the rejection. The response asserts the claimed polypeptide is a type II C type lectin which contains a classical CRD domain at the C-terminus and the requisite amino acids that are known to mediate calcium binding, which is asserted at page 4. This argument has been thoroughly reviewed, but was found unpersuasive. At page 4, the specification teaches that the claimed polypeptide has homology to a number of different C type lectins with different structures and functions. Further, at page 5, the specification asserts that the claimed polypeptide has similar activity to C-type lectin receptors such as mannose binding lectin, mammon binding lectins, and dendritic cell immunoreceptors. In the previous office action, and reiterated above the examiner set forth that these C type lectins belong to different subfamily's of C type lectins which have different structures and functions. As such, one of skill in the art would not be able to determine, based on homology analysis alone, the presently claimed polypeptide would function like. The office action further set forth, that even if the claimed polypeptide was in the type II subfamily, such was not enough to establish a predictable correlation as to the function of the claimed polypeptide. From the teachings of Akimoto, it is apparent that assignment of SEQ ID NO 4 to a particular subfamily does not make apparent the function or specificity of SEQ ID NO 4 as table 3 shows that different type II receptors have different specificities and bind different ligands. For example human H1 binds galactose and N-acetylgalactoseamine while CD23 binds IgE. The response further asserts that the percent similarity between the claimed polypeptide (SEQ ID NO 4) and DCIR (a type II receptor) is 69% and that one of ordinary skill accepts structural homology based on amino acid sequence identity as a credible method of determining the function of the polypeptide and cites the

Heinkoff reference. This argument as well as the reference have been thoroughly reviewed but were found unpersuasive. Firstly, it is noted that SEQ ID NO 4 has 49% identity over a portion of DCIR. Secondly, ~~Heinkoff~~ Henikoff et al teach that as a result of duplications, which can be dispersed and can involve entire coding regions or modules that correspond to folded protein domains, gene products may acquire new specificities, altered recognition properties or modified functions (See abstract). Further, ~~Heinkoff~~ Henikoff et al teach that the accumulation of experimental evidence concerning gene or protein function or protein structure will provide insights that can be used to deduce possible family relations that would not be compelling by sequence comparison alone (see p. 612, sentence bridging col 2 and col 3). Thus Heinkoff et al teach that the although proteins may have similar folded domains, such does not reliably indicate the function or specificity of a protein and that experimental evidence is required to provide such insights. Further, it is clear from such teachings as well as the teachings of Skolnick et al (Skolnick and Fetrow, TIBTECH, Vol. 18; January 2000, pp 34-39) that one of ordinary skill would not, based on the teachings in the art, accept that structural homology based on amino acid identity would provide a reliable method of determining function of a polypeptide. Skolnick teaches that homology analyses alone does not necessarily offer a predictable correlation between structure and function of proteins. Skolnick teaches that sequence based methods for function prediction are inadequate because of the multifunctional nature of proteins (see abstract). Skolnick teaches that both alignment and motif methods are powerful but that recent analysis has demonstrated significant limitations, suggesting that these methods will increasingly fail as the protein-sequence databases become more diverse (p. 34, col. 2).

The response further asserts that applicants submitted a ClustalW multiple sequence alignment in exhibit 2 of the Response dated 7/25/2002, in which SEQ ID NO: 4 was compared to DCIR, providing evidence that the claimed polypeptide is a type II C type lectin receptor. This argument, as well as exhibit 2 of the 7/25/2002 response have been thoroughly reviewed but were found unpersuasive. Firstly, it is noted that the specification does not limit analysis of protein homology of SEQ ID NO: 4 to DCIR, but also to other C lectin proteins. The specification teaches that SEQ ID NO: 4 also has 39% identity and 55% similarity with "mouse macrophage C-type lectin. However, these sequences are not identical, and Balch et al teach the unpredictability with regard to amino acid changes in this protein. Balch teaches that comparative sequence analysis suggests that "mouse macrophage C-type lectin" (referenced as mMCL by Balch) has carbohydrate binding capabilities, but that little can be postulated about the ability or specificity of mMCL from its protein sequence alone because even within a relatively small, conserved domain, binding specificity can be altered with the mutation of only one or two amino acids (see Iobst and Drickamer, JBC, 1994, vol. 269, pp 15512-15519). Balch further teaches that some molecules containing C-type lectin domains have been shown to bind peptide sequences such that this versatility makes predicting putative ligands for this type of lectin domain difficult, and that for mMCL this task is even more challenging because a serine rather than the typically conserved proline separates the two critical sugar binding residues corresponding to Glu-185 and Asn-187 (p. 18662, paragraph bridging col. 1 and 2). With regard to DCIR, as stated in the previous office action, and reiterated above, Bates et al (J. Immunol., 1999, vol. 162, pp 1973-1983) teach that dendritic cell immunoreceptor (DCIR) is a type II membrane glycoprotein (abstract) and that it is of potential importance in regulation of dendritic

cell function, *however it's function or activity in such regulation is not taught*. Bates teaches that the Ca2+ ligating residues are well conserved in DCIR, displaying closest homology with hepatic ASG-PR, but that localization of the gene on chromosome 17 could suggest that DCIR represents an evolutionary intermediate between the NK cell receptors and the hepatic lectins [different type II lectins - see Akimoto et al] and that the cytoplasmic domain of DCIR contains one ITIM motif which is present in the cytoplasmic tail of C-type lectin like molecules expressed by NK cells (p. 1979, col. 2, last two sentences of last full para; and bridging para pp 1979-1980). Thus, it is clear that the prior art does not support a predictable structure function correlation with regard to type II, C type lectins. Additionally, Fetrow teaches (Fetrow et al., J. Mol. Biol., vol. 282, pp 703-711, 1998) that although function prediction by homology to previously characterized proteins is extremely successful and is fast, cheap and reliable, there are several problems that limit its potential utility, one of which is that sequence homology does not guarantee functional similarity (p 704, col. 1, 1st full paragraph). Fetrow teaches that "threading"(analysis using structure prediction tools) can identify topological cousins, that is, protein families such as the barrels with similar structures, but dissimilar functions. Fetrow teaches using a three dimensional descriptor of the active site of a protein, termed "fuzzy functional form" (FFF) and argues that threading alone is not enough to provide the required information about function because it has been shown that pairs of proteins can have similar structures but unrelated functions (p. 706, col. 2, last para). Fetrow teaches that because such topological cousins exist, knowledge of the structure is not equivalent to identification of protein function. Although the alignment studies provided by applicant indicate that the claimed polypeptide contain domains common to type II C type lectins and that the claimed polypeptide

likely belongs to the C type lectin family, such alignment is not sufficient to indicate to the ordinary artisan the specific function or biological activity of the claimed polypeptides, so that the ordinary artisan would know how to use the claimed invention. The art specifically teaches, that sequence alignment alone is does not necessarily provide a predictable correlation between the structure and specific function of a protein.

The response further asserts that SEQ ID NO: 4 has a CRD with calcium binding sites and can bind mannose and glucose and that therefore the polypeptide of SEQ ID NO 4 retains the carbohydrate binding capability and can bind mannose and glucose as ligands in a calcium dependent manner. This argument has been thoroughly reviewed but was not found persuasive. Firstly, assuming that SEQ ID NO: 4 can bind mannose and glucose in a calcium dependent manner, such analysis was not provided in the specification as filed. Furthermore, the specification does not assert a utility for SEQ ID NO: 4 with regard to such. Secondly, as this information was not present in the application as filed, nor was it known in the prior art, such is not evidence of a well established utility for SEQ ID NO: 4. In addition, such utility is not considered specific as it can be applied to a broad class of proteins (type II, C type lectins, which have different functions and specificities as evidenced by applicant's cited reference, Drickamer and Taylor, Annu. Rev. Cell. Biol. Vol. 9, pp 237-264, see pp 241-243) and is not considered substantial because it does not indicate a predictable, immediately apparent function for the polypeptide of SEQ ID NO: 4, without further experimentation. The response asserts that SEQ ID NO: 4 is expected to have a similar utility as DCIR in that it may play a role in DC maturation and serve to uptake antigens. This argument was not found persuasive to overcome the rejection because the specification has not demonstrated such, and, as discussed above, the

homology analysis of SEQ ID NO: 4 with DCIR does not provide a predictable correlation as to the function of SEQ ID NO: 4.

The response asserts that since SEQ ID NO: 4 shares 69% similarity with DCIR, one of skill would expect that the expression patterns would be similar. This argument was thoroughly reviewed but was not found persuasive as the examiner could find no teaching in the art that proteins with similarity in amino acid composition are generally accepted to have similar expression patterns and the response provided no reference to such. Further, it is clear from the teachings of Drickamer and Taylor (cited by applicants) that type II C type lectins are not necessarily expressed in the same cells. For example, Drickamer and Taylor teach that mammalian asialoglycoprotein receptor is found on hepatocytes while Kupffer cell receptor is found on Kupffer cells (macrophages of the liver) and lymphocyte receptors are found on the surfaces of lymphocytes. With regard to the putative uses of SEQ ID NO: 4 on page 6, it is noted that the specification does not assert or provide evidence that SEQ ID NO: 4 regulates the migration of DCs and their subsequent interaction in lymphoid cells or that SEQ ID NO: 4 modulates the inflammatory response and is used as a therapeutic for allergic airway diseases such as asthma. Further, the reference cited by Wang et al, with regard to a showing that soluble lectins were able to suppress, in a dose dependent manner, allergen stimulated bronchial inflammation associated with asthma, such does not represent a well established utility at the time the specification was filed (reference published in 2001).

For these reasons, and the reasons made of record above, and in the previous office action, the rejection of claims 10-11, 20 and 31 under 35 USC 101 and 112/first paragraph is maintained.

Conclusion

5. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

6. No claims are allowable.

7. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Souaya whose telephone number is (703) 308-6565. The examiner can normally be reached Monday-Friday from 9:00 AM to 6:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Jones, can be reached on (703) 308-1152. The fax phone number for this Group is (703) 305-3014.

Any inquiry of a general nature should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Jehanne Souaya
Primary Examiner
Art Unit 1634

Jehanne Souaya
July 30, 2003